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Comments on Sulimani et al.: A weekly 35,000 IU vitamin D supplementation improves bone turnover markers in vitamin D deficient Saudi adolescent females

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Dear Editor,

In a recent paper, Sulimani et al. examined the short-term effects of weekly 35,000 IU vitamin D supplementation on bone turnover markers. The study included 68 vitamin D deficient girls aged 13–19 years from intermediate and secondary schools in Riyadh (Saudi Arabia). All subjects were given 35,000 units of cholecalciferol drops every week for 4 weeks. Blood samples were taken at baseline and after 4 weeks to determine circulating levels of 25(OH)vitamin D, parathyroid hormone (PTH), calcium phosphorus, alkaline phosphatase, osteocalcin and carboxy-terminal telopeptides of crosslinks of type I collagen (β CTX). They found a significant increase in serum 25(OH) vitamin D, a decrease in circulating levels of alkaline phosphatase, osteocalcin, β CTX and PTH levels [1].

Three aspects need to be highlighted.

First, the authors reported that intake within the range of ~ 600 to ~ 1000 IU of vitamin D3 daily is capable of reaching ≥ 50 nmol/l in more than 97% of adolescents. In this context, a short-term and a long-term comparison of the dose chosen by the authors (~ 5000 IU of vitamin D3 daily) with the standard dosage would be of clinical interest. Hence, the results obtained by Sulimani et al. should encourage the design of clinical trials focused on this issue [1].

Second, the authors did not report if potential causes of malabsorption were ruled out. In particular, celiac disease (CD) is a chronic, immune-mediated disorder, characterized by small intestinal malabsorption of nutrients, after the ingestion of wheat gluten or related proteins from rye and barley, in genetically susceptible individuals [2]. Patients with CD could present with signs and/or symptoms not directly referable to the gastrointestinal tract. Extraintestinal features, including osteopenic bone disease, may remain undiagnosed for years and can lead to loss of bone mineral density [3]. Osteopenic bone disease is the result of impaired calcium absorption by the affected intestine, vitamin D deficiency caused by impaired absorption of vitamin D and binding of intraluminal calcium and magnesium to unabsorbed dietary fatty acids to form insoluble soaps. With prolonged calcium malabsorption, patients may develop secondary hyperparathyroidism, resulting in mobilization of calcium from bones and further exacerbation of osteopenia. To highlight the relevance of the relationship between CD and osteopenia, we have reported that up to 58% of adults at first diagnosis of CD suffered from osteopenia [4]. In the study by Sulimani et al. the eventual measurement of anti-tissue transglutaminase antibody or anti-endomysium antibody in these adolescents is not reported [1]. This could enrich their results.

Finally, although statistically significant, changes in some parameter remained inside the normal range (β CTX from 0.52 $\mu\text{g/l}$ to 0.32 $\mu\text{g/l}$; PTH from 6.3 pmol/l to 3.5 pmol/l ; calcium from 2.25 mmol/l to 2.38 mmol/l , phosphorous from 1.27 mmol/l to 1.24 mmol/l); in any case, beyond the mere modification of the biochemical parameters, data about their clinical consequences would be of interest (i.e. data about changes in Dual-energy X-ray absorptiometry, DXA or the Fracture Risk Assessment Tool, FRAX). [1]. Hence, further studies should clarify these issues.

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